51 Episodic Neurologic Symptoms

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Definition

Intermittent neurologic symptoms comprise a group of complaints that may be associated with dysfunction of many organ systems, including the central nervous system, cardiovascular system, and vestibular apparatus. Intermittent metabolic disturbances and psychiatric problems may also result in neurologic symptoms. Despite these diverse etiologies, all the conditions discussed here share the following features: (1) They are intermittent; (2) they are recurrent; (3) they are usually brief, lasting minutes to hours; (4) the patient is usually asymptomatic between attacks; and (5) the symptoms are usually stereotyped for an individual patient.

Although intermittent neurologic symptoms often have a benign prognosis, some may be a manifestation of a serious condition. Multiple sclerosis, myasthenia gravis, and certain other neurologic illnesses may have intermittent symptoms. Most patients with these conditions have a more chronic course on which intermittent symptoms are superimposed, however; they will not be discussed further here.

Technique

Many conditions resulting in episodic neurologic symptoms are discussed in other chapters, and this chapter is intended as a general approach to a patient with these symptoms. Special problems include migrainous events versus partial seizures versus transient ischemic attacks (TIAs); syncope versus seizures; evaluation of dizziness; and evaluation of ill-defined symptoms.

When evaluating a patient with intermittent neurologic symptoms, it is reassuring that a careful approach to the problem usually results in a definite diagnosis, or at least a few reasonable hypotheses, which can be further evaluated. It is useful to remember the clinical axiom that "common things are common" and that seizures, syncope, TIAs, labyrinthine disorders, and hyperventilation are seen frequently in everyday practice. The most common clinical situation is for the patient to present for medical care because of the symptom itself. Occasionally, intermittent neurologic symptoms are discovered incidentally on review of systems in a patient presenting with an unrelated problem. With these points in mind, certain investigational strategies are helpful.

First, it is important to try to classify the patient's major symptom into one of the categories in Table 51.1, since this immediately narrows the diagnostic possibilities. This is accomplished by listening carefully to the patient's chief complaint. The most important things to determine are whether the patient had any focal neurologic symptoms or any alteration of consciousness. The history is of critical importance because the examination between events is often normal. It is always best to listen attentively to the patient tell the story in his or her own words, rather than ask specific questions too early. Invaluable diagnostic clues are usually identified by a careful listener. The patient should be encouraged to "start at the beginning" and relate the first symptom, then others in temporal sequence. Information about onset is very important, and if not volunteered, should be sought by direct questioning. It is very helpful to ask about precipitating situations or activities through such questions as these: "Is there any way you can tell a spell will occur?" or "Do you get any warnings?" or "Is there any way you can bring on a spell?" While the most obvious value of such questions is to detect the focal onset (aura) of seizures, the questions may also provide information about stimulus-precipitated seizures, exercise-induced syncope, vasovagal syncope, or cataplectic attacks caused by surprise or emotion. The patient should be asked how long it takes for symptoms to reach maximum intensity, and how long the entire event lasts. It is helpful to ask whether the patient has ever experienced such symptoms in the past. Some symptoms may recur intermittently over many years, including those caused by migraine, many kinds of seizures, or vasovagal syncope. Recent onset of symptoms may add a sense of clinical urgency in patients with syncope of cardiac origin or with transient ischemic attacks. A change in character or frequency of attacks suggests a progressive lesion, such as a cerebral neoplasm causing a changing seizure disorder. Associated symptoms may be critically important, but may require further questions: "Do you notice anything else?" or "Do you feel sick in any other way?" For example, a patient with Ménière's disease may notice ear fullness,

Table 51.1
Classification of Episodic Neurologic Symptoms

Major symptom	Causes
Focal neurologic dysfunction	Transient ischemic attacks Migrainous events Partial seizures
Alteration of consciousness	Seizures (primary generalized, secondarily generalized, or partial complex) Syncope Sleep disturbances (narcolepsy, sleep apnea) Drug reactions
Dizziness	Labyrinthine disturbances Vertebrobasilar transient ischemic attacks Presyncope Psychiatric etiologies
Miscellaneous symptoms	Psychiatric etiologies (panic attacks, hyperventilation syndrome, pseudoseizures, anxiety, depression) Rare syndromes (pheochromocytoma, arrhythmias, carcinoid syndrome, hypoglycemia, etc.) Alteration of muscle tone (atonic or myoclonic seizures, cataplexy) Drug reactions

tinnitus, or hearing loss associated with vertigo; a patient with a migrainous neurologic deficit may notice a headache and nausea as the deficit improves. Further details of each of these associated symptoms should be sought in the order they are noticed.

If there is a question in the examiner's mind whether or not focal neurologic symptoms occurred, a review of neurologic symptoms should be undertaken. The patient should be quizzed as to whether the event was accompanied by any visual disturbance, numbness, weakness, clumsiness, or speech disturbance. If any such symptoms occurred, it should be determined if they "spread" to other areas and the rate of such spread. The patient should be questioned about a "postictal" period of unresponsiveness or confusion. In many cases, diagnostically crucial information regarding the spells must be obtained from a friend or family member. At this phase of the interview, it is usually apparent into which of the major categories the patient's symptoms fit, and it is appropriate to ask more detailed questions to differentiate among entities in the appropriate category.

Basic Science

The basic pathophysiology of syncope, epilepsy, transient ischemic attacks, migraine, and sleep disturbance is discussed in other chapters of this book, but a few points should be emphasized.

Transient ischemic attacks probably result from transient interruption of blood flow to a specific portion of the brain. There is reasonable evidence that most TIAs are due to embolization of platelet and fibrin debris from atherosclerotic plaques in the proximal portions of the large cerebral vessels, especially at the carotid bifurcation. This phenomenon can sometimes actually be visualized in retinal arteries in patients with amaurosis fugax. Bright cholesterol emboli or platelet-fibrin debris may be seen at the bifurcations of these vessels, which are branches of the ophthalmic artery, which in turn arises from the internal carotid. With time, these microemboli break up and pass distally. The concept of arterial laminar flow, whereby blood flowing along the arterial wall adjacent to a plaque is always distributed to the same area of brain tissue, is thought to explain the stereotyped nature of recurrent TIAs. It is important to understand, however, that there are many other causes of TIAs. For example, cardiac abnormalities of many types may result in cerebral emboli. Cerebral vasculitis caused by systemic lupus erythematosus, giant cell arteritis, or granulomatous angiitis may interrupt blood flow. Patients with thrombocytosis, erthyrocytosis, or leukocytosis may have arterial or capillary sludging with transient ischemic symptoms. Rarely, patients with multiple large vessel atherosclerotic occlusions may have tenuous collateral blood flow to certain areas of the brain, with transient ischemia caused by a drop in systemic blood pressure.

While it is generally accepted that seizures are due to abnormal spontaneous neuronal depolarization and spread of electrical activity, there are many etiologies. In primary generalized epilepsy, the abnormal discharges appear simultaneously in all head regions and may originate in a centrally located generator with diffuse projections, perhaps the thalamic reticular system. Impaired consciousness may be the first manifestation, and if motor signs occur, they are bilateral. Examples include primary tonic-clonic seizures, true petit mal seizures, and certain rarer types. The age of onset of this type of seizure is usually under 20 years. There is a strong hereditary predisposition, and most patients experience no warning or aura. Acquired epilepsy may occur at any age and may result from such varied insults as birth hypoxia, head trauma, brain tumors, or cerebrovascular accidents. In these cases, the abnormal electrical activity begins locally, often resulting in focal neurologic symptoms. These partial seizures may spread to become secondarily generalized, resulting in a tonic-clonic seizure. A focal seizure always implies focal brain dysfunction and should focus attention on questions and diagnostic tests designed to find the cause. The older the patient at onset of seizures, the more likely a clear etiology can be found. In patients over age 50, as many as 20% of patients with onset of seizures have a cerebral neoplasm. It should always be remembered that there are many toxic and metabolic causes for seizures, including acute hypoxia, hypoglycemia, hyperosmolar states, hypo or hypernatremia, hypocalcemia, uremia, and drug or alcohol withdrawal. History, examination, and lab studies should be directed at uncovering such etiologies in patients with a first seizure.

Migraine may be defined as a paroxysmal disturbance of cephalic neurovascular function, presenting as episodic headaches that may be associated with autonomic, visual, or neurologic symptoms of variable prominence. The pathophysiology of migraine is still not well understood. The widely held belief that the neurologic and visual events are caused by cerebral vasoconstriction and the subsequent headache result from vasodilation is certainly too simplistic. There are no clear data to support the hypothesis that migrainous neurologic events are due to vasoconstriction, and some researchers believe they are a cortical phenomenon of unknown etiology. Although cerebral blood flow is decreased during migrainous neurologic dysfunction, it is unclear if this is a primary event or a secondary event coupled to neuronal dysfunction.

Syncope is due to global cerebral hypoperfusion. The causes are therefore almost exclusively cardiovascular, and examination and laboratory studies should focus on causes of hypotension and decreased cardiac output, especially bradyarrhythmias and tachyarrhythmias. A neurologic cause is found in 10% or fewer patients with syncope, usually an unusual or unwitnessed seizure. Multiple high-grade vascular stenoses or transient increased intracranial pressure are very rare causes of syncope. Syncope should not be attributed to cerebrovascular disease unless accompanied by focal neurologic symptoms. Often, no clear etiology for syncope can be found, in which case a benign course is usually seen.

The symptoms seen in the narcolepsy syndrome are due to disordered sleep cycles causing inappropriate involuntary sleep during the day. Rapid eye movement (REM) sleep normally occurs after an orderly progression of sleep to progressively deeper stages, a process that takes 2 to 3 hours. Persons with narcolepsy often progress directly into REM sleep with concomitant dreaming and loss of muscle tone. This is thought to explain the phenomena of sleep paralysis and hypnagogic hallucinations. The loss of muscle tone during cataplectic attacks may also be an REM-related event. Patients with sleep apnea may have airway obstruction in the oropharynx, loss of central regulation of respiration, or both. They are chronically sleep deprived, since they invariably develop apnea as they reach deeper sleep stages, resulting in multiple brief nighttime arousals, which the patient does not recall. Consequently, the patient has involuntary episodes of daytime sleep.

Vertigo results when there is disturbance in the normal

balance between the left and right vestibular systems and their respective brainstem connections. Acute loss of function or overactivity of one vestibular organ results in vertigo. Horizonal nystagmus is a frequent clinical accompaniment due to extensive connections between the vestibular nuclei and eye movement systems. Patients with vertigo on a "central" basis almost invariably have other brainstem symptoms and findings due to close proximity of the vestibular nuclei to other brainstem nuclei, as well as ascending and descending sensory and motor tracts.

Brief episodes of global cerebral hypoperfusion may result in presyncope, manifested by lightheadedness and other symptoms. Patients with chronic, ill-defined dizziness should be considered for possible psychiatric causes provided that ataxia, posterior column dysfunction, and drug or medication side effects have been excluded.

New data suggest that panic attacks may result from dysfunction of central autonomic regulation, for which there may be a familial predisposition. Since lactate and bicarbonate infusions sometimes reproduce symptoms in susceptible persons, it is also possible that a reduction in free ionized calcium causes symptoms. These findings have opened new possibilities for research and treatment. The causes of conversion reactions are best explained on a psychodynamic basis.

Clinical Significance

Patients with episodic neurologic symptoms may present a major diagnostic challenge. Sometimes symptoms are caused by underlying medical or neurologic conditions that require immediate treatment; on other occasions, episodic symptoms are nonspecific events or a manifestation of a psychiatric disturbance. With such a wide range of different illnesses producing similar symptoms, it is not surprising that the conscientious clinician can sometimes be baffled and frustrated. The problem is compounded by the fact that many patients have normal physical examinations. At times, it may be difficult to decide between initiating a lengthy and expensive diagnostic evaluation and merely reassuring the patient and providing careful clinical follow-up. Even when exhaustive diagnostic evaluations are undertaken, the etiology may not be found. Observation is preferable to empirical treatment.

The most common causes of episodic focal neurologic dysfunction are transient ischemic attacks (TIAs), migrainous phenomena, and partial seizures. While each of these entities has certain identifying characteristics (Table 51.2), in some circumstances diagnostic problems may occur. TIAs are "negative" neurologic events (characterized by loss of function), whereas partial seizures are "positive" or excitatory phenomena. Migrainous events are often "positive" (flickering lights) when they involve visual phenomena and "negative" (weakness, numbness) when other neurologic symptoms occur.

Transient ischemic attacks usually do not present a diagnostic problem, except for confusion with migraine or when symptoms are vague or nonspecific. It is safest to define TIAs as a focal neurologic deficit that can be clearly localized to a specific cerebral vascular distribution. TIAs usually begin suddenly, reach maximum deficit quickly, and have no precipitating or aggravating factors. Although TIAs are defined as lasting less than 24 hours, most are less than 15 minutes in duration. Most TIAs occur in older individuals with atherosclerotic risk factors, especially hypertension. Symptoms of carotid TIAs include unilateral weakness or sensory disturbance, aphasia, unilateral neglect, or amaurosis fugax (fleeting blindness in one eye). Vertebrobasilar symptoms include vertigo, diplopia, dysarthria, homonymous hemianopsia, total (cortical) blindness, ataxia, and shifting or bilateral weakness or numbness. Between 12 and 40% of patients with TIAs will eventually suffer a completed stroke, with the greatest risk in the first few weeks. Patients with TIAs occurring in rapid succession (crescendo TIAs) are especially at risk.

Migraine poses a special problem in the diagnosis of episodic symptoms because its manifestations are so protean. When a characteristic headache is part of the attack, the diagnosis is simple. However, headache may be inconspicuous or absent. Clinically, it is important to understand that migrainous neurologic events usually begin insidiously and spread gradually over minutes, whereas TIAs and focal seizures are much more precipitous in onset. The most common migrainous events other than headache are visual, especially scintillating scotomas. The patient may describe these

Table 51.2
Features of Common Episodic Focal Neurologic Symptoms

	Partial seizures	TIAs	Migrainous events
Onset	Sudden	Sudden	Usually gradual
Nature of event	"Positive"	"Negative"	Usually positive, occasionally negative
Spread of symptoms	Rapid, if occurs (seconds)	Rapid, if occurs (seconds–minutes)	Usually occurs, always gradual (minutes)
Total duration of spell	Usually less than 5 minutes	Usually less than 15 minutes	Usually 15 to 25 minutes
Age at first symptoms	Variable	Over age 40, usually over age 60	Under age 40; usually adolescent or young adulthood
Constancy of symptoms	Onset stereotyped, but may "spread" or progress to generalized seizure	Usually stereotyped	Often stereotyped, but may change to involve new symptoms over time
Long-term course	Usually chronic, nonprogressive, except with mass lesions	Variable, may disappear; one-third have stroke	Chronic, fluctuating
Special identifying features	Underlying focal brain abnormality identified by history, examination, or diagnostic tests	Atherosclerotic risk factors, concurrent heart disease, or underlying rare predisposing illness (hematologic, collagen vascular); abnormal arterial radiologic studies	Usually followed by vascular headache; neurologic symptoms may be dissociated from headache; family history of headache

as flickering lights that gradually spread across the visual field, often taking the form of a jagged, irregular line ("fortification phenomena"). Other visual events, such as photomas (cloudlike scotomas) or homonymous hemianopsia, may occur. Occasionally, patients with migraine may experience spreading numbness, weakness, aphasia, or even brainstem symptoms. Headache usually begins in 20 to 30 minutes as these symptoms subside. Although onset is usually before middle age, migraine can change over time in terms of frequency and manifestations. It has been postulated that some older patients with what appear to be TIAs may actually have symptoms due to migraine. The diagnosis of a "late-life migraine accompaniment" should be made only when the symptoms exhibit a typical slow migraine "build-up," and there is no other identifiable cause. The patient should be closely questioned about a past history of intermittent throbbing headaches and about a family history of headache.

There are many kinds of *partial seizures*, but the pattern is usually stereotyped for the individual patient. Simple partial seizures consist of isolated motor, sensory, or visual symptoms that usually occur paroxysmally (without warning), build in intensity over seconds, and either disappear or progress to secondary generalization of the seizure discharge, resulting in a major motor seizure. It is therefore very important to ask if convulsions or episodes of loss of consciousness ever follow a focal neurologic symptom. Examples of symptoms seen in focal seizures include jerking of one side of the face, one arm, or one leg, which may rapidly "spread" to other areas. Focal sensory seizures may also occur, with spread in a similar fashion. Visual seizures may consist of simple geometric shapes when the focus is in the occipital region or complex visual phenomena, such as a scene or vision, when the focus is in the temporal lobe. At times, there is alteration of perception of visual stimuli such as macropsia (items appear magnified) or micropsia (items appear small). Other examples of partial seizures involving the temporal lobe include unusual odors (uncinate hallucinations), noises, or psychic phenomena, which may be simple (an emotion such as fear or a recurrent thought) or complex (déjà vu). Such symptoms are easily confused with psychiatric conditions. The intensity of the events and their stereotyped and paroxysmal nature should be helpful in identifying the events as partial seizures. Complex partial seizures (psychomotor seizures) are a very common form of partial seizures and often begin with one of the "temporal lobe" phenomena described above, progressing to clouding of consciousness and purposeless "automatic" motor activity of the face or limbs. Since partial seizures are usually acquired and suggest an underlying focal brain lesion, it is important to question the patient about a history of head injury, central nervous system infection, stroke, or any progressive neurologic symptoms suggestive of a cerebral neoplasm.

Intermittent loss of consciousness immediately suggests *syncope* or *generalized seizures* (Table 51.3). The best way to distinguish between these etiologies is to observe a spell. Since this is seldom possible, the physician must often rely on information from family or friends. The patient's own history of the event is also important, but may be incomplete. With a few exceptions (true petit mal in childhood), motor activity is prominent in seizures associated with loss of consciousness. Tonic-clonic seizures, especially if witnessed, are usually easily diagnosed. During major motor seizures, some patients will be incontinent, bite their tongue or cheek, or suffer other injuries. Most patients with syncope are limp, with no associated motor movements, although occasionally a few clonic jerks may be noticed. Rarely, a tonic–clonic seizure occurs. Preceding symptoms are extremely important. Patients with partial seizures that secondarily generalize may have an "aura" consisting of a specific neurologic symptom prior to loss of consciousness. Most patients with primary generalized epilepsy have no warning at all, however. Before a syncopal attack, many patients experience lightheadedness, weakness, blurred vision, or nausea. The patient should always be questioned regarding precipitating factors. Seizures are usually paroxysmal, but occasionally are precipitated by photic stimulation (strobe lights, watching television), hyperventilation, sleep deprivation, or drug or alcohol withdrawal. Some seizures occur

Table 51.3
Features of Common Causes of Episodic Alterations of Consciousness

	Generalized seizures	Syncope	Sleep disturbances
Onset	Sudden	Variable (seconds–minutes)	Usually gradual (minutes)
Warning	No, except aura with seizures that secondarily generalize	Yes; dizziness, diaphoresis, nausea, blurred vision	Yes; sleepiness
Total duration of spell	1 to 2 minutes	10 seconds to 5 minutes	5 to 10 minutes
Postictal state	Yes	No	No
Age at first symptoms	Under 20 for primary generalized seizures; variable for partial seizures that secondarily generalize	Variable, depends on etiology	Under age 30 for narcolepsy, over age 50 for sleep apnea
Long-term course	Usually chronic, nonprogressive, except when caused by mass lesions	Variable, depends on etiology	Chronic
Special identifying features	Motor activity, cyanosis, tongue biting, incontinence; usually paroxysmal, sometimes focal neurologic symptoms if begins focally	Usually no motor activity, pale, diaphoresis; look carefully for precipitating activities and underlying systemic or cardiovascular disease	Sleepiness usually occurs when sitting or engaged in repetitive activities; may have episodes of "microsleep" with automatisms; cataplexy and other symptoms in narcolepsy, restless nighttime sleep with snoring in sleep apnea; consider drug-induced causes, other rare syndromes

only when falling asleep, during sleep, or when awakening. Syncope may be associated with similar or identical precipitating factors for each episode. Examples include assuming an erect posture (orthostatic hypotension) and increased vagal tone (cough, urination, or emotional event). Associated palpitations, chest pain, dyspnea, diaphoresis, or nausea should be sought. A history of bradyarrhythmias or tachyarrhythmias, ischemic heart disease, or syncope associated with exertion raises suspicion of syncope of cardiovascular origin. Almost all patients with major motor seizures will be unresponsive for a number of minutes during the seizure, followed by a period of disorientation and confusion that may last for hours. Patients may complain of muscle soreness and tenderness. Although patients with syncope may complain of feeling weak, they usually are quickly aware of their surroundings after a brief loss of consciousness lasting seconds or minutes.

Although listening carefully to the story related by the patient and witnesses will usually differentiate between syncope and seizures, at times the distinction is very difficult. In this situation, the patient may need a diagnostic evaluation for both possibilities. An electroencephalogram is sometimes helpful but is frequently normal interictally in patients with known seizure disorders. If no clear diagnosis can be established, the patient should be followed carefully for possible clues that may later permit accurate classification. It is better to underdiagnose epilepsy in uncertain cases, considering the medical and social consequences the diagnosis carries.

Sleep disturbances are a more uncommon cause of episodic altered consciousness. Patients with narcolepsy or sleep apnea have irresistible daytime sleepiness, often at inopportune or dangerous times. Patients will admit to falling asleep during conversation, while driving a car, and occasionally even when active. Some patients have episodes of "microsleep" during which they may perform semipurposeful acts for which they have no recall. These automatisms result in confusion with partial complex seizures. *Narcolepsy* usually begins under age 30, and is almost always associated with cataplexy, a generalized or focal loss of muscle tone that occurs with strong emotion. Patients should be asked if they have ever collapsed suddenly when frightened, angry, or in the midst of laughter. Less common symptoms associated with the narcolepsy syndrome are sleep paralysis (transient total paralysis while falling asleep or on awakening) and hypnagogic hallucinations (very realistic nightmares that occur as the person falls asleep). Although patients with *sleep apnea* have excessive daytime sleepiness, they lack the other components of the narcolepsy syndrome. The sleeping partner will frequently give a history of nighttime snoring, restless sleep, and frequent episodes of apnea during sleep. Polysomnography will help confirm the diagnosis in difficult cases. Patients with episodic alteration or loss of consciousness should also be questioned about prescription, over-the-counter, or illicit drugs that might be causing their symptoms.

Dizziness is a common and often confusing complaint, meaning different things to different people. It is especially critical to ascertain if the patient has a sensation of movement, since vertigo is usually the result of a labyrinthine disorder. Associated symptoms of tinnitus or hearing loss also suggest a "peripheral" labyrinthine problem. Most patients with acute vertigo prefer to lie still, since minimal movement exacerbates their discomfort. Nausea, vomiting, and ataxia often occur. Among the many causes of recurrent "peripheral" vertigo are Ménière's disease, vestibular neuronitis, and benign positional vertigo. Vertigo is occasionally associated with TIAs involving the cerebellum or brainstem. By generally accepted convention, however, isolated vertigo should not be considered due to brainstem dysfunction unless other brainstem symptoms or signs are present. The cause of nonspecific dizziness or "fuzzy-headedness" is often difficult to elucidate. Anxious patients may have a feeling of floating, detachment, or lightheadedness that is described as dizziness and may be associated with chronic hyperventilation. The onset is usually gradual, and the feeling often lasts for long periods of time. Patients with presyncope may complain of lightheadedness, which sometimes progresses to syncope. Finally, patients with posterior column disturbance or peripheral neuropathy may complain of dizziness, which is better characterized by unsteadiness, or "dizziness in the feet."

Patients with conversion reactions, hyperventilation syndrome, panic attacks, or depression may complain of complex neurologic symptoms. In addition to dizziness, patients may complain of intermittent sensory symptoms (tingling or numbness), weakness, or memory loss. Patients with *conversion symptoms* can usually be identified by the dramatic and sometimes bizarre nature of their intermittent symptoms. The history is often related with surprising detachment and unconcern called "la belle indifference." The patient usually has no insight into the psychiatric basis of the symptoms or the unusual nonphysiologic findings that often are present on the neurologic examination. Many patients have a previous history of conversion reactions, and a few patients have Briquet's syndrome, a history of multiple conversion reactions involving multiple organ systems over time, and often a history of multiple surgical procedures by well-meaning surgeons. The *acute hyperventilation syndrome* is usually recognized by anxiety, acro-oral paresthesias, a feeling of "not being able to take a deep enough breath," and, in its most severe form, carpal-pedal spasm. At times, the symptoms can be reproduced by asking the patient to hyperventilate for several minutes. Some patients with *panic attacks* can identify uncomfortable situations that precipitate their spells, but most cannot. Patients usually complain of severe apprehension, dizziness, chest discomfort, palpitations, or fear of "losing control." The onset is usually under age 40, the course is chronic and fluctuating, and many patients also have phobias. Occasionally patients with what appear to be panic attacks are actually having paroxysmal tachycardia, or autonomic dysfunction associated with pheochromocytoma, hypoglycemia, or complex partial seizures. Neurologic symptoms also occur in depressed patients, but are often more constant than episodic. Nondescript symptoms such as tiredness, weakness, or dizziness predominate.

Pseudoseizures are episodes that appear to be epileptic seizures, but have a psychiatric origin. They can often be distinguished from true seizures by their atypical motor activity, lack of postictal state, and associated "secondary gain." Occasionally, only combined EEG and videotelemetry during an attack will distinguish between true seizures and pseudoseizures, and some patients have both. Other psychiatric events that may be episodic include "temper tantrums" and psychogenic amnesia. Questions directed at the patient's general adaptation, social functioning, and previous history of psychiatric difficulties are also helpful in separating patients with symptoms of psychiatric etiology from those due to other causes.

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